

Aggression Among Combat Veterans: Relationships With Combat Exposure and Symptoms of Posttraumatic Stress Disorder, Dysphoria, and Anxiety

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Prior research has revealed heightened aggressive behavior among veterans with PTSD. This study tested a model examining the interrelationships among combat exposure, posttraumatic stress disorder (PTSD) symptoms, dysphoric symptoms, and anxiety symptoms in predicting aggressive behavior in a sample of 265 male combat veterans seeking diagnostic assessment of PTSD. Combat exposure was indirectly associated with aggression primarily through its relationship with PTSD symptoms. Symptoms of PTSD were directly related to aggression, and indirectly related to aggression through dysphoric symptoms. Results highlight the role of PTSD symptoms and dysphoric symptoms with respect to aggressive behavior among this population, and suggest the relevance of aggression theory to the study of combat veterans.

Studies have consistently shown that male veterans with posttraumatic stress disorder (PTSD) evidence higher rates of violent outbursts and aggressive behavior than those without the disorder, and exhibit more hostility expression and poorer anger control (Beckham, Feldman, Kirby, Hertzberg, & Moore, 1997; Kulka et al., 1990; Lasko, Gurvits, Kuhne, Orr, & Pittman, 1994; McFall, Fontana, Raskind, & Rosenheck, 1999). Unfortunately,

very little research has attempted to elucidate potential pathways through which PTSD symptomatology may lead to higher levels of aggression, or tested conceptual models for aggression among this population (Beckham, Moore, & Reynolds, 2000). Work in this area may serve to inform aggression-control interventions for combat veterans suffering from PTSD (Chemtob, Novaco, Hamada, & Gross, 1997). The current study tested a model for aggressive

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behavior among a sample of combat veterans receiving clinical services, with PTSD symptoms hypothesized as a central explanatory variable, and combat exposure and associated psychological symptoms (dysphoric and anxiety symptoms) posited as additional explanatory variables.

Studies of veterans demonstrate a positive relationship between combat exposure and measures of aggression (Byrne & Riggs, 1996). It has been suggested that combat exposure is associated with aggression partly due to the reinforcement and modeling of violence in the military (Gimbel & Booth, 1994), and some researchers have found that combat exposure and PTSD symptoms have independent effects on aggressive behavior (Beckham et al., 1997). However, several studies indicate that combat exposure is associated with aggression primarily through its relationship with PTSD symptoms (Byrne & Riggs, 1996; Orcutt, King, & King, 2003).

Posttraumatic stress disorder is highly comorbid with several psychiatric disorders among veterans, particularly mood disorders and other anxiety disorders, and veterans with PTSD evidence higher depression and anxiety severity than those without the disorder (e.g., Orsillo et al., 1996). Most research in this area has been cross-sectional, and the developmental trajectories of psychiatric symptoms are largely unknown. However, some investigations indicate that PTSD typically precedes affective disorders (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), and PTSD symptoms more strongly prospectively predict the development of depressive symptoms than does depression predict PTSD symptoms (Erickson, Wolfe, King, King, & Sharkansky, 2001).

There is some evidence to suggest that PTSD comorbidity partially accounts for the relationship between PTSD and aggressive behavior. Taft et al. (2005), in a recent examination of data from the nationally representative National Vietnam Veterans Readjustment Study (Kulka et al., 1990), demonstrated that the presence of comorbid depression was among the strongest risk factors for partner violence perpetration among veterans with PTSD. Aggression theory, particularly the work of Berkowitz (1990), also suggests that negative affect variables may help explain the relationship between PTSD symptoms and aggression.

Berkowitz' cognitive-neoassociationistic model holds that negative affect is connected with anger-related feelings, thoughts, memories, and aggressive inclinations in associative networks, and the experience of negative affect activates the entire network. Therefore, those who experience more frequent and severe negative affect also experience heightened feelings, thoughts, and memories related to anger, and have a higher propensity for aggressive behavior. Several empirical investigations have provided support for the basic tenets of this model (see Berkowitz, 1990; Verona, Patrick, & Lang, 2002), and a number of studies demonstrate relationships between measures reflecting negative affect and measures of aggression (Bjork, Dougherty, & Moeller, 1998; Maiuro, Cahn, Vitaliano, Wagner, & Zegree, 1988; Mammen, Kolko, & Pilkonis, 2002; Pan, Neidig, & O'Leary, 1994; Waltz, Babcock, Jacobson, & Gottman, 2000).

The following hypotheses were tested using a structural equation modeling (SEM) framework: (a) combat exposure would be related to higher aggression both directly and through its relationship with PTSD symptoms; (b) PTSD symptoms would be indirectly associated with aggression through dysphoric and anxiety symptoms; and (c) in addition to its indirect effects, PTSD symptoms would exhibit a direct association with aggression when accounting for the other predictors, due to other (unmeasured) potential mediators, such as physiological reactivity and cognitive factors (see Beckham et al., 2000).

METHOD

Participants

Participants included 265 male combat veterans who sought a diagnostic assessment for PTSD at the VA Boston Healthcare System between September 1999 and September 2003. Of the 273 potential participants, 8 were excluded from the present investigation because they did not report experiencing combat on the Combat Exposure Scale (Keane et al., 1989). At the time of the assessment, 81% of the veterans were either applying for disability status or a disability upgrade within the VA system.

Eighty-one percent of study participants were White, 13% were African American, 3% were Hispanic, 2% were Asian or Pacific Islander, and 1% were American Indian or Alaskan Native. One percent of the participants reported that their race or ethnicity fit none of these categories. On average, veterans were 53.9 years old ($SD = 9.2$ years). Slightly over one half (51%) of the participants were married, 28% were divorced, 11% were never married, 6% were separated, 3% had a live-in relationship partner, and 1% were widowed. With respect to annual income, 32% reported earning between \$0 and \$10,000, 14% earned \$10,001 to \$20,000, 21% earned \$20,001 to \$30,000, 10% earned \$30,001 to \$40,000, 12% earned \$40,001 to \$50,000, and 11% earned over \$50,000.

Most participants (79%) reported that the majority of their service occurred during the Vietnam War. The remaining participants were veterans of World War II (7%), Operation Desert Storm (7%), and the Korean War (4%). Three percent of the participants reported service during other war eras. Vietnam veterans and other combat veterans did not significantly differ in their reports of combat exposure, $t(249) = -1.27, ns$. Participants represented all branches of the active duty military: Army (61%); Marines (21%); Navy (11%); and Air Force (8%). The majority of participants (70%) had enlisted in the military, whereas 24% were drafted and 6% had volunteered for the draft. Details of military service were routinely verified during diagnostic assessments by inspection of veterans' military records (e.g., DD-214 form).

Measures

The following measures were examined in the current investigation and were drawn from a larger battery of instruments administered to all veterans seeking a PTSD-focused clinical assessment at this site.

Combat exposure was assessed using the Combat Exposure Scale (CES; Keane et al., 1989). The CES is a 7-item self-report instrument designed to measure veterans' experience of combat-related war stressors.

Posttraumatic stress disorder symptoms were examined using the Clinician-Administered PTSD Scale (CAPS;

Blake et al., 1990, 1995). The CAPS is a widely used semistructured interview based on the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV; American Psychiatric Association, 1994) criteria that also has commonly been employed to derive a continuous measure of PTSD symptomatology (see Weathers, Keane, & Davidson, 2001). Interviewers assign frequency and intensity scores to each of the 17 PTSD symptoms on a 5-point Likert scale based on the previous month. A total severity score for each symptom is computed by summing the frequency and intensity scores. The internal consistency reliability estimate for the CAPS in the present investigation was .88. Sixty-eight percent of the current sample met criteria for PTSD according to the original CAPS scoring rule described by Blake et al. (1990), which requires symptom frequency to be >1 and symptom intensity to be >2 for a positive endorsement of a particular PTSD symptom.

Dysphoric symptoms were assessed using the Beck Depression Inventory (BDI; Beck et al., 1961). The BDI is a 21-item self-report measure that is widely used to assess the attitudes and symptoms of depression and dysphoria among clinical and normal populations. For each item, respondents are asked to choose which of four statements best describes how they have been feeling in the 2 weeks prior to the assessment. The internal consistency reliability estimate for the BDI was .92.

Anxiety symptoms were measured with the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988). The BAI is a 21-item self-report measure designed to assess anxiety symptoms distinct from those of depression. Responses to each item are based on how much the respondent was bothered by the symptom in the past week, and range from 0 (*not at all*) to 3 (*severely*). The internal consistency reliability estimate for the BAI was .94.

Aggression was examined using a 6-item measure originally developed for a multisite trial initiated under the auspices of the Cooperative Studies Program of the Department of Veterans' Affairs that examined the utility of psychophysiological measures in predicting the presence or absence of PTSD (see Keane et al., 1998). This measure assessed instances of aggressive behavior that ranged

from verbal abuse and threats of violence (e.g., *was verbally abusive, threatened someone with a weapon*) to actual perpetration of violence (e.g., *had a physical fight with someone; used a weapon against someone*). This measure was significantly predicted by age, PTSD hyperarousal and avoidance/numbing symptoms, physiological reactivity to trauma cues, and alcohol problems in a recent investigation of a large clinical sample of male Vietnam veterans (Taft et al., 2006), attesting to its construct validity among the population of interest. Moreover, in two separate structural equation modeling (SEM) models, the predictors accounted for approximately 30% of the variance in this aggression outcome. Responses for this measure were given on a 7-point scale (0 = *never*; 1 = *once*; 2 = *3 to 5 times*; 4 = *6 to 10 times*; 5 = *11 to 20 times*; 6 = *more than 20 times*) and were referenced to aggressive incidents towards anyone in the 4 months prior to assessment. All items were recoded to reflect the estimated frequency of the behavior (*never* = a score of 0; *once* = a score of 1; *3 to 5 times* = a score of 4; *6 to 10 times* = a score of 8; *11 to 20 times* = a score of 15; *more than 20 times* = a score of 25) and summed to compute a total aggression frequency score. Such methods to obtain estimates of frequency scores are commonly used for measures of aggression (see Straus, 1990). Ratings of aggressive behavior referred to the prior 4 months.

Data Analyses

First, descriptive statistics were computed based on the composite variables. Next, bivariate correlations were computed among all study variables, and effect sizes corresponding to these associations were interpreted in terms of suggestions made by Cohen (1988). We next employed SEM to test the hypothesized interrelationships among study variables. This methodology is especially useful for examining complex associations among multiple constructs. A measurement model incorporating the latent variables and their manifest indicators was first specified and evaluated, followed by structural models intended to evaluate hypotheses concerning the direct and indirect ef-

fects relating combat exposure, PTSD symptoms, dysphoric symptoms, anxiety symptoms, and aggression.

For all SEM analyses, matrices of covariances were submitted to the LISREL 8 program (Jöreskog & Sörbom, 1993). The full information maximum likelihood (FIML) estimator was used to accommodate missing data under the assumption that data were missing at random (Little & Reuben, 1987). Maximum likelihood estimation was used. Covariances among residuals were always fixed at 0. A square root transformation was applied to the aggression parcels to correct for positive skew.

RESULTS

Descriptive Statistics

Table 1 presents descriptive statistics for the composite variables. Participants reported perpetrating an estimated average of nine aggressive behaviors in the 4 months prior to assessment. The PTSD symptom scores were slightly lower than those found in studies of combat veterans with PTSD (Elhai, Frueh, Davis, Jacobs, & Hamner, 2003). On average, participants endorsed moderate to severe levels of both dysphoric and anxiety symptoms (Beck & Steer, 1993; Beck, Steer, & Garbin, 1988). CES scores indicate that participants averaged moderate levels of combat (Keane et al., 1989).

Table 2 presents bivariate correlations between all study variables. Posttraumatic stress disorder symptoms, dysphoric symptoms, and anxiety symptoms were significantly associated with aggression, with associations falling within the medium range. Combat exposure was not significantly

Table 1. Descriptive Statistics for Study Variables

Variable	<i>M</i>	<i>SD</i>	Range
Combat exposure	22.83	10.22	1–41
PTSD symptoms	72.41	27.16	6–136
Dysphoria symptoms	24.18	12.00	0–58
Anxiety symptoms	22.84	12.76	0–63
Aggression	8.70	13.83	0–113

Note. PTSD = posttraumatic stress disorder.

Table 2. Bivariate Correlations Among Study Variables

Variable	1	2	3	4	5
1. Combat exposure	—				
2. PTSD symptoms	.27*	—			
3. Dysphoric symptoms	.12	.39*	—		
4. Anxiety symptoms	.11	.32*	.68*	—	
5. Aggression	.10	.29*	.35*	.26*	—

Note. PTSD = posttraumatic stress disorder.

* $p < .05$.

associated with aggression at the bivariate level. Both dysphoric and anxiety symptoms were significantly associated with PTSD symptoms, with medium effect sizes obtained. A medium-sized association was also found between combat exposure and PTSD symptoms. Although the correlation between dysphoric and anxiety symptoms was high, this association falls below widely accepted thresholds for both multicollinearity and poor discriminant validity (Kline, 2005), and is consistent with relationships found in other studies (e.g., Brown, Chorpita, & Barlow, 1998). This amount of overlap suggests that the two latent variables share slightly less than 50% of their variance, leaving the other half of the variance in each unaccounted for by the other.

Structural Equation Modeling

Measurement model. The measurement model was comprised of five constructs or latent variables and their 11 manifest indicators. The combat exposure measure was composed of a single causal indicator representing the sum of scores on the seven items included in the Combat Exposure Scale (Keane et al., 1989). The PTSD symptom latent variable had four manifest indicators from the CAPS (Blake et al., 1990) derived from a factor analysis of the measure (King, Leskin, King, & Weathers, 1998). Specifically, indicators addressed symptoms of reexperiencing (five items), avoidance (two items), emotional numbing (five items), and hyperarousal (five items). The dysphoric symptoms latent variable was composed of three manifest indicators based on a factor analysis of the BDI (Beck et al., 1961) by Tanaka and Huba (1984). The first indicator (12 items)

reflected negative cognitions. The second indicator (six items) reflected dysphoria-related somatic symptoms. The third indicator (three items) reflected physiological manifestations of dysphoria. For the anxiety symptoms latent variable, one 12-item and one 9-item indicator were derived from the Beck Anxiety Inventory (Beck et al., 1988). These item sets addressed somatic and cognitive aspects of anxiety, respectively, and were based on a prior factor analysis by Beck and his colleagues (1988). The sum of aggressive acts included in the aggression measure was treated as a single causal indicator.

Manifest indicators were specified to load on designated latent variables and the model was fit to the data. The resulting measurement model provided a reasonably good fit to the data, $\chi^2(36) = 96.52$, $p < .001$; the Akaike information criterion (AIC; Akaike, 1987) was 156.52; the corrected Akaike information criterion (CAIC; Bozdogan, 1987) was 293.11; the root mean square error of approximation (RMSEA; Steiger, 1990) was .08, with a 90% confidence interval (CI) of .061–.10; the comparative fit index (CFI; Bentler, 1990) was .95; and Steiger's corrected form (Steiger, 1990) of the goodness-of-fit index (GFI; Jöreskog & Sörbom, 1993) was .96. Importantly, the RMSEA met the .08 standard of good fit (Browne & Cudeck, 1993), and the CFI met the recommended minimum value for good fit (CFI = .95; Bentler, 1990). Moreover, all factor loadings were significant, as indicated by corresponding critical ratios exceeding 2.00 (Jöreskog & Sörbom, 1993).

Structural model. We next turned our attention to examining relationships among the five latent variables using the fit associated with the measurement model (equivalent to a fully saturated model in which all variables are allowed to covary) as the base for judging more parsimonious models. The specification of paths was guided by the hypotheses involving the direct and indirect influences of earlier variables on downstream latent variables. An initial model with seven structural coefficients was specified. Based on our hypotheses, we specified paths between combat exposure and both PTSD symptoms and aggression, between PTSD symptoms and both dysphoric and anxiety symptoms, as well as aggression, and between the dysphoric

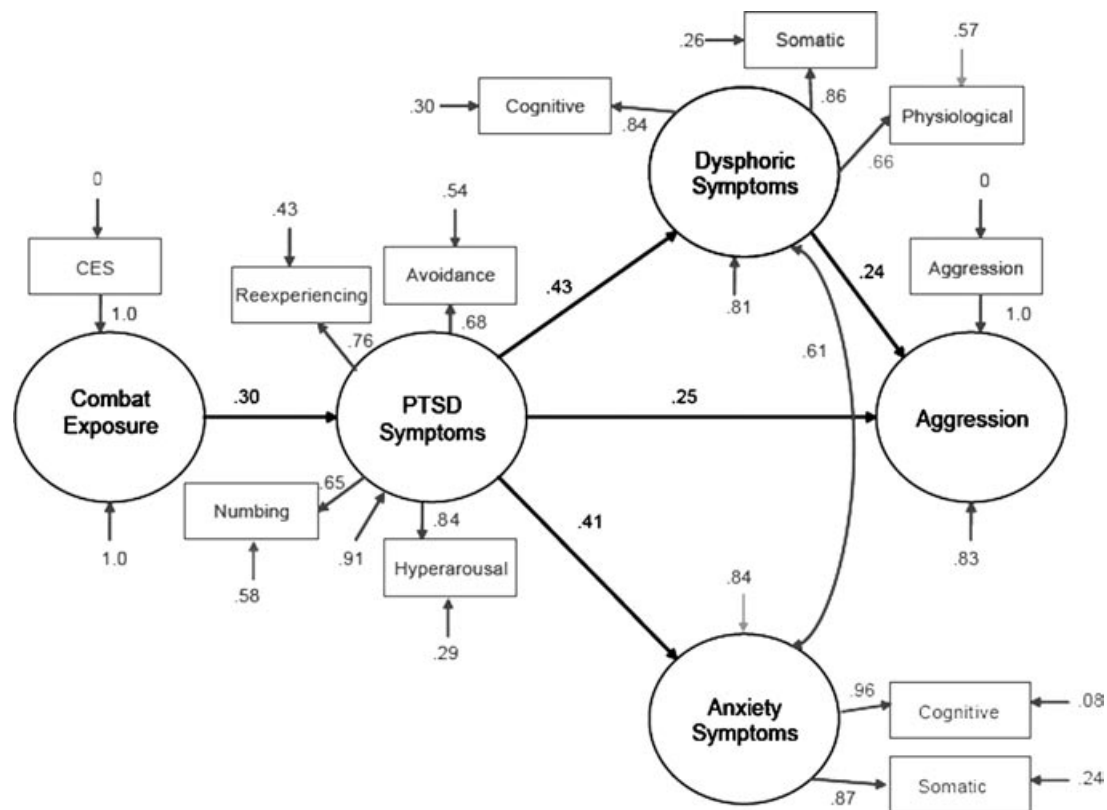


Figure 1. Final model incorporating both measurement and structural modeling results. Path coefficients are standardized. All paths specified within this model were significant, as indicated by critical ratios >2.00 (Jöreskog & Sörbom, 1993).

and anxiety symptom variables and aggression. The dysphoric and anxiety symptom variables were allowed to covary.

The lack of a significant difference in chi squares between this model and the base model (i.e., the fully saturated measurement model) suggested that reducing the model in this way did not damage model-to-data fit, $\Delta\chi^2(2) = 0.47$, *ns*. However, an examination of this model suggested the further deletion of two additional paths that demonstrated nonsignificant partial relationships (i.e., corresponding to critical ratios less than 2.00; Jöreskog & Sörbom, 1993). Specifically, we deleted the paths from combat exposure to aggression and from anxiety symptoms to aggression. We arrived at a final model of best fit, $\chi^2(40) = 97.06$, $p < .001$, depicted in Figure 1. The RMSEA for this model (.07 with a 90% CI of .056–.093)

met the .08 standard of good fit (Browne & Cudeck, 1993). The comparative fit index (CFI; Bentler, 1990) was .95, meeting the recommended minimum value for good fit. The finding of a nonsignificant chi-square statistic difference between this more constrained model and the initially hypothesized model ($\Delta\chi^2(2) = 0.07$, *ns*) further indicates that this more parsimonious model provided good fit to the data. Evidence for this model is also provided by the finding that values for the AIC, CAIC, and RMSEA decreased and were smallest for this model relative to the preceding models (i.e., the fully saturated model and the initially hypothesized model), given that smaller values indicate closer fit (Akaike, 1987; Bozdogan, 1987; Hu & Bentler, 1998).

Consistent with recent recommendations for testing mediation (MacKinnon, Lockwood, Hoffman, West, &

Table 3. Standardized Effects for Final Model

	Direct effects	Indirect effects	Total effects
Combat exposure			
Combat exposure to PTSD	.30 (4.44)		.30 (4.44)
Combat exposure to dysphoric symptoms		.13 (3.70)	.13 (3.70)
Combat exposure to anxiety symptoms		.12 (3.64)	.12 (3.64)
Combat exposure to aggression		.11 (3.53)	.11 (3.53)
PTSD Symptoms			
PTSD Symptoms to dysphoric symptoms	.43 (5.69)		.43 (5.69)
PTSD Symptoms to anxiety symptoms	.41 (5.49)		.41 (5.49)
PTSD Symptoms to aggression	.25 (3.30)	.11 (3.00)	.35 (5.13)
Dysphoric symptoms			
Dysphoric symptoms to aggression	.24 (3.37)		.24 (3.37)

Note. Critical ratios (i.e., *Z* scores) are included in parentheses; all effects achieved statistical significance.

Sheets, 2002), we evaluated evidence for indirect effects in terms of the significance of the product of coefficients representing the association between the predictor and the mediator and the association between the mediator and the outcome. Within LISREL, these tests are based on the Sobel test (Baron & Kenny, 1986; Sobel, 1982). We found significant indirect associations of PTSD symptoms to aggression through dysphoric symptoms and from combat exposure to aggression through PTSD symptoms and dysphoric symptoms, providing additional support for the final model. Table 3 provides standardized estimates of all direct, indirect, and total effects.

DISCUSSION

The current study tested a model examining the interrelationships among combat exposure, PTSD symptoms, dysphoric symptoms, anxiety symptoms, and aggressive behavior. As hypothesized, PTSD symptoms were directly associated with higher levels of aggression when accounting for the other variables of interest in the structural model. Only dysphoric symptoms appeared to partially account for the effects of PTSD symptoms on this outcome. Contrary to expectations, combat exposure was not directly associated with aggression in the structural model, though this factor exerted an indirect effect on aggression through PTSD symptoms.

The absence of a direct relationship between combat exposure and aggression is in contrast to some prior research (Beckham et al., 1997). This finding is also inconsistent with conceptualizations of aggression that emphasize its reinforcement, acceptance, and modeling in the military or its adaptive nature during combat situations (Gimbel & Booth, 1994; Taft et al., 2005). Results suggest that combat exposure influences aggressive behavior primarily through its relationship with PTSD symptomatology, consistent with the results of other recent studies that have similarly tested direct and indirect relationships among these variables (Byrne & Riggs, 1996; Orcutt et al., 2003).

Findings suggest that dysphoric symptoms in particular serve as a mechanism for the relationship between PTSD symptoms and aggression, supporting previous work suggesting that depressive symptomatology is a risk factor for aggression among veterans with PTSD (Taft et al., 2005). Anxiety symptoms were not associated with aggression when considered together with the other variables of interest in SEM analyses. Differences between dysphoric and anxiety symptoms were not expected in the current investigation, though general aggression theory has emphasized the role of dysphoric affect (Berkowitz, 1990). Among those experiencing PTSD symptoms, dysphoric symptoms may be particularly likely to be associated with disinhibitory influences, which override internal and external restraints that would otherwise limit aggressive responses (Chemtob et al., 1997).

Findings from this study have potentially important clinical implications. As results from several previous studies suggest (Beckham et al., 1997; Jordan et al., 1992), treatment of PTSD symptoms may lead to a reduction in aggressive behavior among combat veterans. According to Berkowitz' (1990) cognitive-neoassociationistic model, the experience of dysphoric affect is likely to activate associative networks of anger-related feelings, thoughts, memories, and aggressive inclinations, making aggressive behavior more likely. This theoretical model and data from the current study suggest that the identification and treatment of dysphoric symptomatology should also potentially be incorporated into anger and aggression management interventions for combat veterans experiencing symptoms of PTSD.

This preliminary study demonstrates a need for the application of existing aggression theory in models for PTSD and aggression. According to Berkowitz (1989, 1990) and other aggression theorists (Dodge & Crick, 1990; Lazarus, Averill, & Opton, 1970), in addition to the experience of negative affect, bodily reactions and higher-order cognitive processing influence the expression of aggression. Attributional processes and prior learning play an important role in most conceptualizations of aggression and may be particularly salient for combat veterans suffering from symptoms of PTSD. For these individuals, anger-related thoughts and hostile attributions are likely to be heightened due to prior experiences of trauma, fear, and life-threat, and a hypersensitivity to potential threats in the environment (Chemtob et al., 1997; Novaco & Chemtob, 1998). Future research is needed to examine the cognitive and information-processing systems that accompany PTSD symptoms and differentiate key emotional experiences to enhance our understanding of the full associative network leading to aggression among this population.

Directionality cannot be assumed in this study. For example, aggression perpetration may cause increased dysphoric symptoms, particularly among individuals high in combat-related PTSD who experience more guilt related to their aggression. The retrospective and self-report nature of our measurement devices also bears consideration.

Memories of combat experiences may have changed with the passing of time and this change may have differentially occurred as a function of PTSD symptoms and negative affect (King & King, 1991). Participants seeking compensation may have exaggerated their reports of combat exposure, psychopathology, and negative affect, and/or aggression, inflating associations. The compensation-seeking status of many participants also limits the ability to generalize current findings to the larger population of veterans not seeking compensation or care from the Department of Veterans' Affairs healthcare system.

Future research should utilize a broader, multi-method assessment approach that assesses state negative affect and that uses laboratory designs to investigate the tenets of aggression theory among military veterans. In addition, future investigations that examine a broader array of aggressive behaviors are needed, as are studies that can represent latent variables with multiple measures of key constructs. Diagnostic measures of PTSD with measured interrater reliability should also be used in future studies. Models for aggression among veterans should incorporate variables reflecting premilitary functioning and prior trauma. Alcohol use is another potentially key variable, given previously established links between alcohol variables and both PTSD symptoms and aggression (e.g., Savarese, Suvak, King, & King, 2001).

Despite these limitations, this study represents an early step in better understanding aggressive behavior among this population. Findings indicated that PTSD symptoms largely account for the influence of combat exposure on aggressive behavior, and dysphoric symptoms partially explain the relationship between PTSD symptoms and aggression. Further research in this area may serve to mitigate the myriad interpersonal and health consequences associated with such maladaptive behavior.

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